



**Today's webinar:**

# **Diagnosis and Management of Post-traumatic Headache**

**May 8, 2014, 1-2:30 p.m. (EDT)**

**Moderator**

**Christian Shenouda, M.D.**

**TBI Physician**

**Contract support to Defense and Veterans Brain Injury Center  
Silver Spring, Md.**





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## **Presenters**

**Jeanne M. Hoffman, Ph.D., ABPP**

**Associate Professor, Department of Rehabilitation Medicine  
University of Washington  
Seattle, Wash.**

**Sylvia Lucas, M.D., Ph.D.**

**Clinical Professor, Department of Neurology, Neurological Surgery and Rehabilitation Medicine  
University of Washington,  
Seattle, Wash.**



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January 16, 2014, 1-2:30 p.m. (EST)

Moderator: Donald Marion, M.D., M.Sc.  
Clinical Affairs Senior Advisor  
Defense and Veterans Brain Injury Center  
Silver Spring, Md.

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Name	Size
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- The Q&A pod is monitored during the webinar; questions will be forwarded to presenters for response during the Q&A session.
- Participants may chat with one another during the webinar using the chat pod.
- The chat function will remain open 10 minutes after the conclusion of the webinar.

# Webinar Overview

- Headache is one of the most common persisting symptoms after traumatic brain injury (TBI) across all levels of injury severity.
- Recent research in civilian and military sample populations has improved the understanding of the prevalence and pathogenesis of the problem after TBI. However, little research has yet to be conducted on treatments for post-traumatic headache.
- This webinar will review the current research on post-traumatic headache and how symptoms of headache can assist with diagnosis. Current recommendations for treatment post-traumatic headache will be described.
- At the webinar's conclusion, participants will be able to:
  - Describe the incidence and prevalence of headache after TBI
  - Relate the critical elements for the diagnosis of headache in individuals with TBI using the symptoms of headache for classification purposes
  - Identify and employ current treatment approaches for headache after TBI

# Presenter: Jeanne M. Hoffman, Ph.D., ABPP



Jeanne M. Hoffman, Ph.D., ABPP

- Associate professor in the Department of Rehabilitation Medicine at the University of Washington
- Clinical psychologist who provides patient care on the inpatient rehabilitation unit and outpatient clinic at the University of Washington Medical Center (UWMC)
- Involved in the TBI Model System research projects for 10 years
- Principal Investigator on a Module Project for the TBI Model System examining the natural history of headache
- Principal Investigator on a field-initiated grant from the National Institute on Disability and Rehabilitation Research to extend the natural history of headache project to individuals with mild TBI
- Extensive experience with the design and analysis of intervention programs for individuals with TBI including projects to evaluate the impact of exercise on mood after TBI

# Presenter: Sylvia Lucas, M.D., Ph.D.



Sylvia Lucas, M.D., Ph.D.

- Clinical professor of Neurology, Neurological Surgery and Rehabilitation Medicine at UWMC
- Founder and director of UWMC Headache Clinic
- Recipient of the Wadsworth Clinical Term Professorship in Headache Research and Practice
- Member of the American Academy of Neurology, Washington State Neurological Society, American Headache Society and International Headache Society; member of board of directors of the Headache Cooperative of the Pacific
- Research interests include post-traumatic headache and headache therapeutics
- Published in journals, including “Nature,” “Journal of Neurophysiology,” “Cephalalgia,” “Headache” and “Journal of Neurotrauma”
- Received the Harold Lamport Biomedical Research Prize

# **Diagnosis and Management of Post-traumatic Headache**

**Jeanne M. Hoffman, Ph.D., ABPP**

**Sylvia Lucas, M.D., Ph.D.**

## Disclosures

- The views expressed in this presentation are those of the presenters and do not reflect the official policy of the Defense Department or the U.S. Government.
- The presenters do not intend to discuss the off-label/ investigative (unapproved) use of commercial products or devices.
- Dr. Hoffman has no relevant relationships to disclose.
- Dr. Lucas discloses these relationships:
  - Research support: St. Jude Medical, Inc., Amgen, MAP Pharma/Allergan
  - Advisory boards: Zogenix, MAP Pharma/Allergan, Kineta
  - Honoraria: Zogenix

# Background

Funding sources and research findings presented:

**"The University of Washington (UW) TBI Rehabilitation Model System," National Institute on Disability and Rehabilitation Research, Principal Investigator, Kathleen Bell. Multi-Site Module Project: "The Natural History of Headache after TBI," Primary Investigator, Jeanne Hoffman. Grant number H133A070032.**

**"Natural History of Headache Following Mild Traumatic Brain Injury," National Institute on Disability and Rehabilitation Research, Principal Investigator, Jeanne Hoffman. Grant number H133G090022.**

**Graphs, charts and tables not referenced in this presentation are associated with research study results.**



# Investigators

## UW Investigators

- Kathy Bell, M.D.
- Sureyya Dikmen, Ph.D.
- Jeanne Hoffman, Ph.D., ABPP (PI)
- Sylvia Lucas, M.D., Ph.D.
- Nancy Temkin, Ph.D.

## TBI Model System Investigators

- Cindy Braden, MA, CCC-SLP, Craig Hospital
- Allen Brown, M.D., Mayo Clinic
- Bobby Brunner, M.D., University of Alabama at Birmingham (UAB)
- Ramon Diaz-Arrastia, M.D., Ph.D., University of Texas Southwestern
- Bill Walker, M.D., Virginia Commonwealth University
- Tom Watanabe, M.D., Moss Rehab

# **Research on Post-traumatic Headache**

# Headache (HA) Diagnoses

## Primary HAs

- Migraine
- Tension-type
- Cluster and its relatives
  - Trigeminal autonomic cephalgias
- Other primary HAs
  - Exertional, coital, hypnic, etc.

## Secondary HAs

- Post-traumatic
- Vascular disease
- Abnormal intracranial pressure, neoplasm, etc.
- Substances
- Central nervous system infection
- Metabolic
- Cervicogenic, eyes, sinuses
- Psychiatric HA
- Neuralgias
- Other

# Definition of Post-traumatic Headache (PTH)

International Headache Society (IHS) Classification, 2<sup>nd</sup> Edition – International Classification of Headache Disorders (ICHD II)

- Meet criteria for severity of head injury (e.g., mild vs. moderate to severe TBI)
- HA develops within seven days of head injury or regaining consciousness after injury
- HA resolves within three months after head trauma (meeting criteria for acute PTH)
- Becomes chronic PTH if persists beyond three months
- BUT no distinct clinical presentation or unique signs
- And timeline for development may be problematic

## How PTH is Defined in the Literature

- Any HA temporally related to TBI
  - No specific classification based on symptoms
- Some follow primary HA diagnostic criteria
- Often variable timing for HA latency following injury

## **Incidence and Prevalence of PTH**

- PTH or HA after TBI is one of the most common persisting symptoms after injury

## **Rates in Service Members**

- 40% of those with TBI reported HA within one week of injury
  - An additional 20% reported headache one month later
  - 40% reported HA more than one month after injury (Theeler, Flynn, & Erickson, 2010)
- Of those found to have concussion, 98% also reported PTH (Theeler & Erickson, 2012)
- Others estimate approximately 32% of those with concussion reported having HA in the past month (Hoge, McGurk, Thomas, Cox, Engel, & Castro, 2008)

# Classification in Service Members

- Migraine most frequent in current research
  - Range from 36% of soldiers in a combat brigade reporting migraine HA
  - Migraine 5.4 times more likely after mild TBI
  - Sample highs of 89-95% of those with PTH meeting criteria for migraine-like HA (Erickson, 2011; Theeler, Flynn, & Erickson, 2010, 2012)



## **Rates in Civilian Samples**

- Early research rates range from 30-90% depending on the study (Nicholson & Martelli, 2004; Solomon, 2005)
  - Time post-injury variable
  - Often clinic samples
  
- Prospective studies
  - Range from 65% at one month, 26% at one year (Dikmen, Machamer, Fann, & Temkin, 2010)
  - 100% at time of injury, 30% at one month, 15% at three months (Faux & Sheedy, 2009)
  - 66% within 7-10 days of injury, none at three months post-injury (Lieba-Samal, Platzer, Seidel, Klaschterka, Knopf, & Wober, 2011)

## **Rates in Civilian Samples (continued)**

- Limited research in athletes
  - Up to 85% with sports-related concussion with PTH (Guskiewicz, Weaver, Padua, & Garrett, 2000)
- Evidence of HA becoming persistent
  - 19-22% at one year post-injury (Dikmen, Machamer, Fann, & Temkin, 2010; Lew, Lin, Fuh, Wang, Clark, & Walker, 2006; Whiteneck, Brooks, Mellick, Harrison-Felix, Terrill, & Noble, 2004)

# Natural History of HA Studies

## Studies funded by the National Institute on Disability and Rehabilitation Research

- TBI Model System Module Study – Total N = 452
  - Natural history of HA after moderate to severe TBI
  - Seven centers participated: UW, Mayo Clinic, Craig Hospital, MossRehab, UAB, UTS and VCU
- Field Initiated Program – Total N = 212
  - Natural history of HA after mild TBI
  - UW

## Methods

- Baseline assessment: Moderate to severe TBI cohort prior to discharge from acute inpatient rehabilitation;  
mild cohort within seven days of injury
- 3-, 6-, 12-month follow-up via telephone
- Assess
  - Incidence and prevalence
  - HA characteristics

## **PTH Early After Injury**

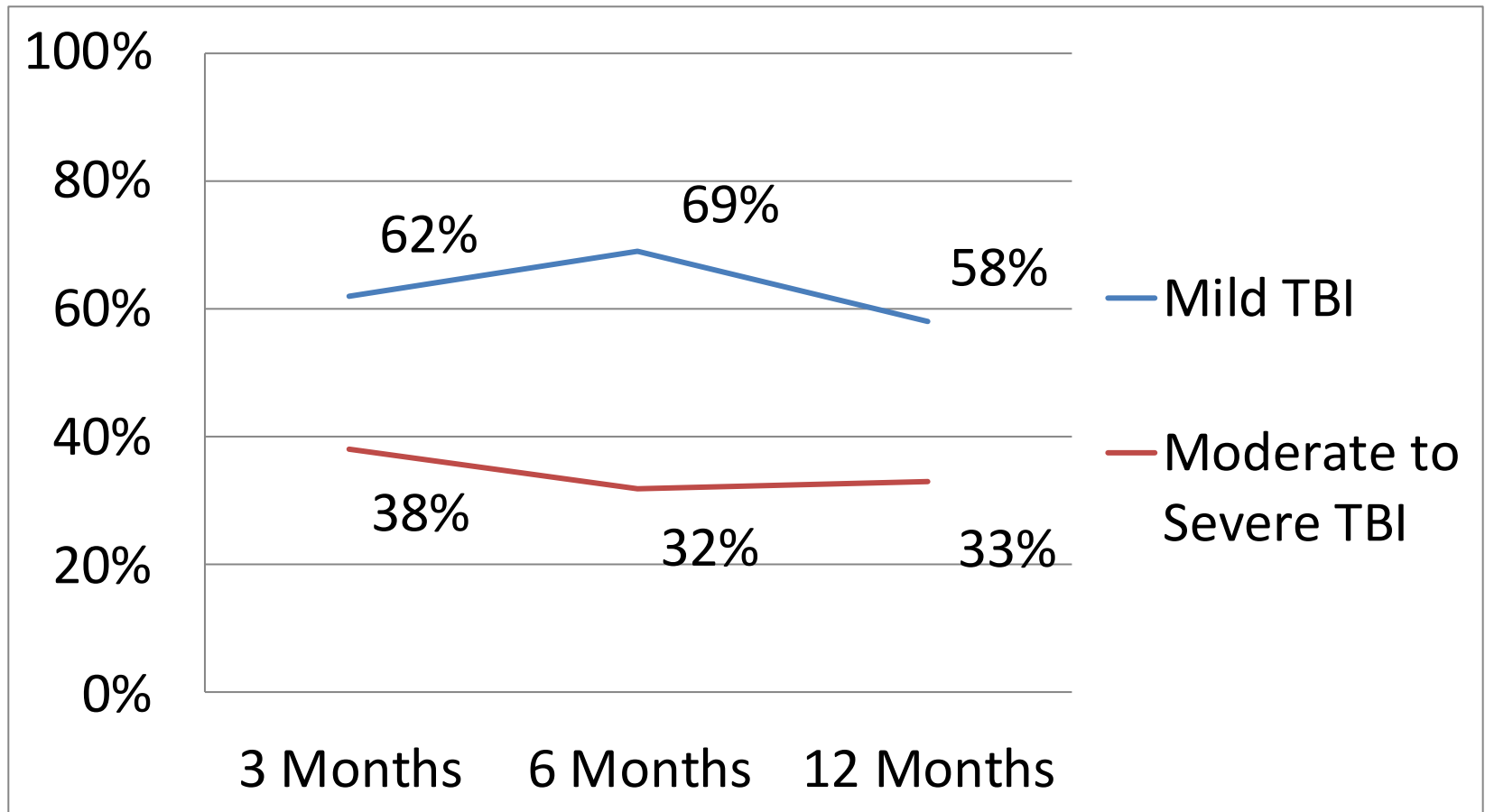
- Moderate to severe TBI
  - 41% assessed during inpatient rehabilitation
  - 71% reported HA across the first year
  
- Mild TBI
  - 54% assessed within seven days of injury
  - 91% reported HA across the first year

## **Comparison of Moderate to Severe TBI to Mild TBI**

- Focus on individuals with NEW or WORSE HA following injury
  - Approximately 18% in both groups had pre-injury HA
- Groups compared at 3, 6, 12 months post injury

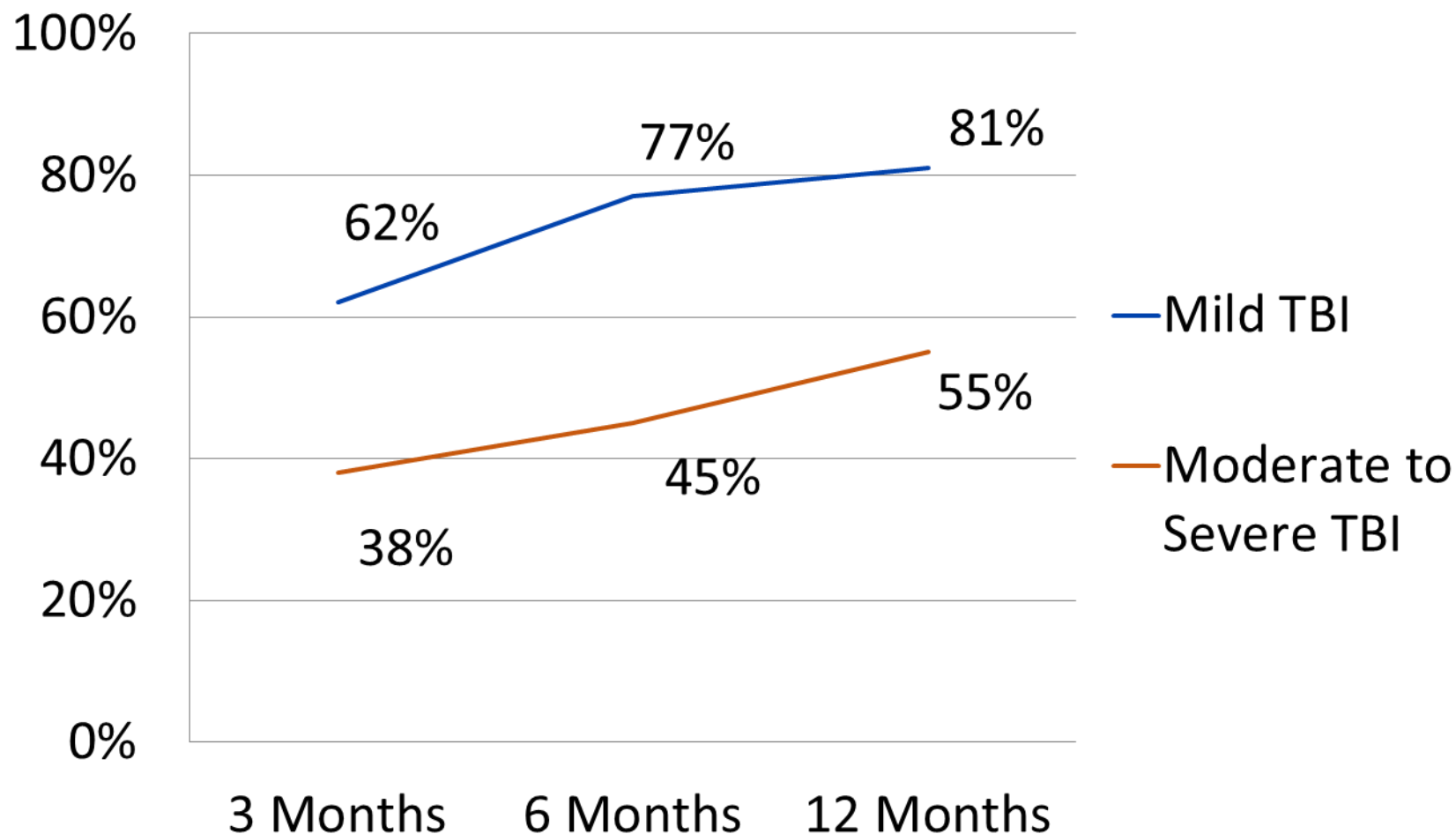
	<b>Moderate to Severe TBI N=403</b>	<b>Mild TBI N=212</b>
Age (years)	42.5	44.4
Male	72%	76%
Race (white)*	75%	75%
High School*	72%	83%
Cause of Injury*		
Vehicle	56%	58%
Assault	9%	5%
Sports	3%	3%
Fall	27%	24%
Hit by Object	2%	2%
Other	3%	8%

## Prevalence of New or Worse HA in the Year after TBI

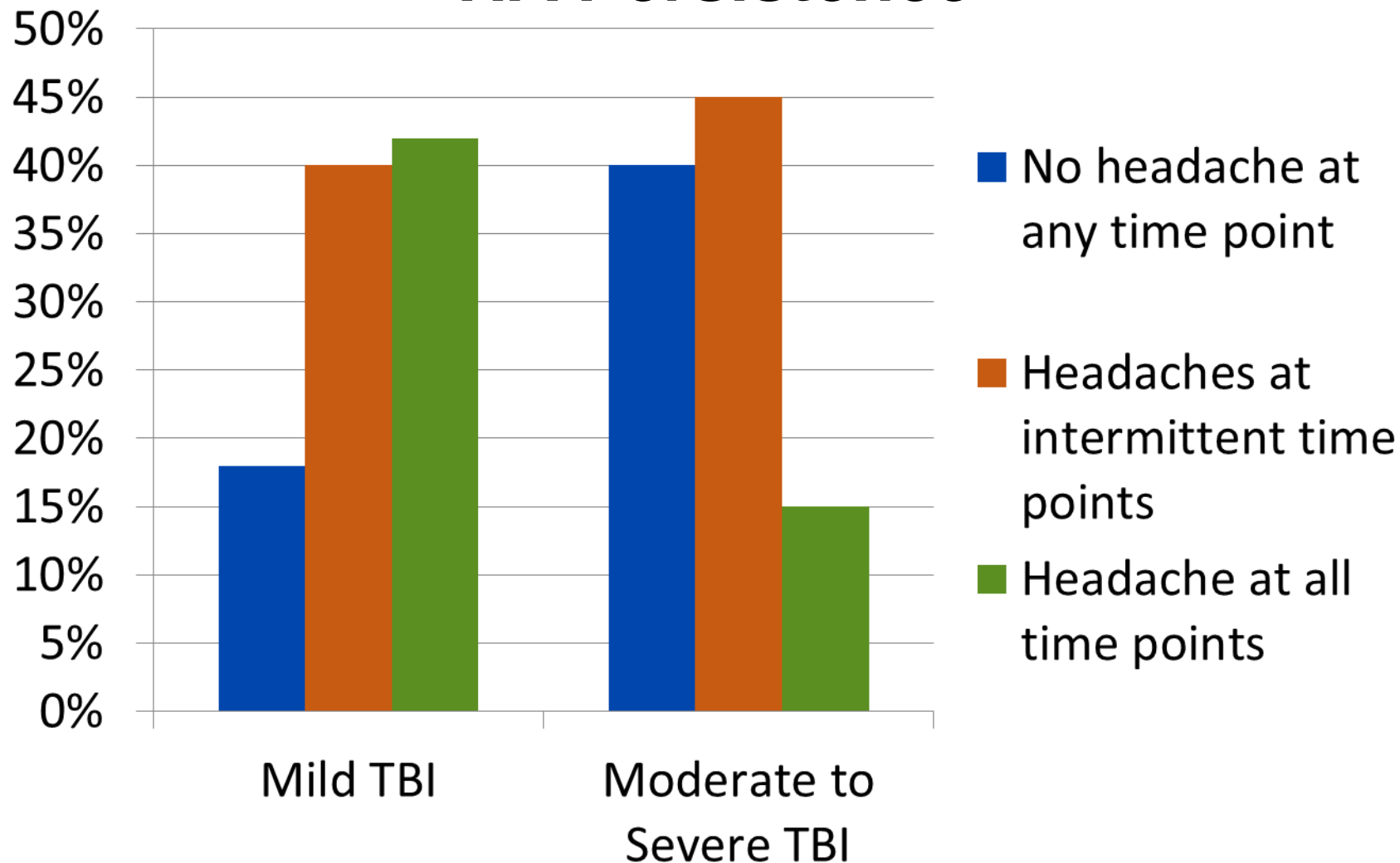




# Cumulative Incidence of New or Worse HA



## HA Persistence



# HA Classification

- Migraine/probable migraine
  - Pain was moderate to severe
  - At least two of the following
    - Significant disabling impact
    - Unilateral
    - Throbbing/pulsating
    - Worsened with movement
    - Either nausea and/or vomiting or sensitivity to light and sound

## **HA Classification (continued)**

- Tension
  - Pain was mild to moderate.
  - Bilateral
  - Vice-like or minimal disabling impact
  
- Cervicogenic
  - Pain was mild to severe.
  - Unilateral
  - Neck pain

## Classification of New or Worse HA

	3 Months		6 Months		12 Months	
	Mod/ Sev	Mild	Mod/ Sev	Mild	Mod/ Sev	Mild
Migraine/ Probable Migraine	60%	49%	59%	49%	61%	49%
Tension	11%	37%	13%	40%	14%	32%
Cervicogenic	5%	4%	3%	4%	4%	4%
Unclassifiable	23%	10%	25%	8%	21%	16%

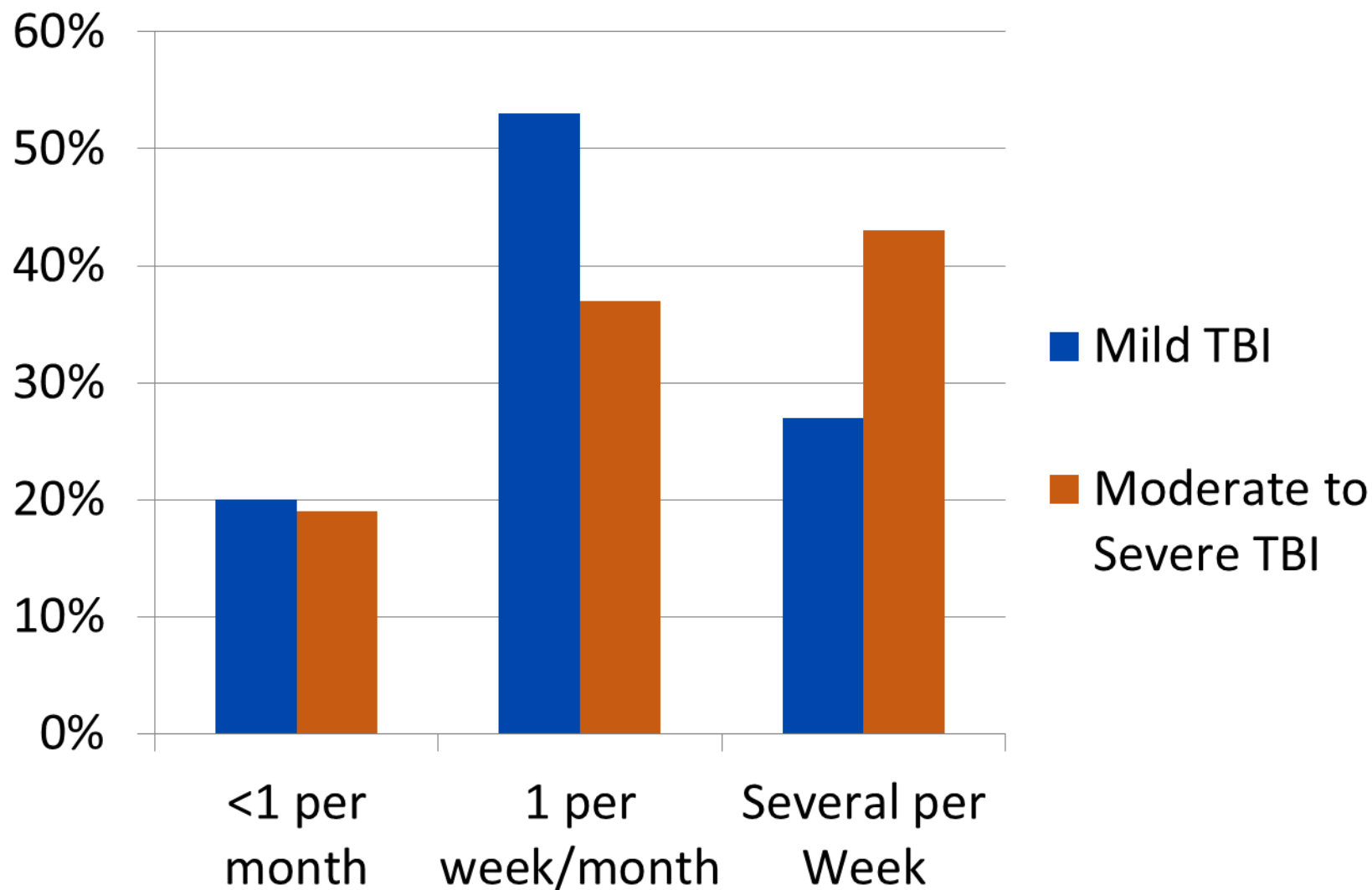
## Classification (continued)

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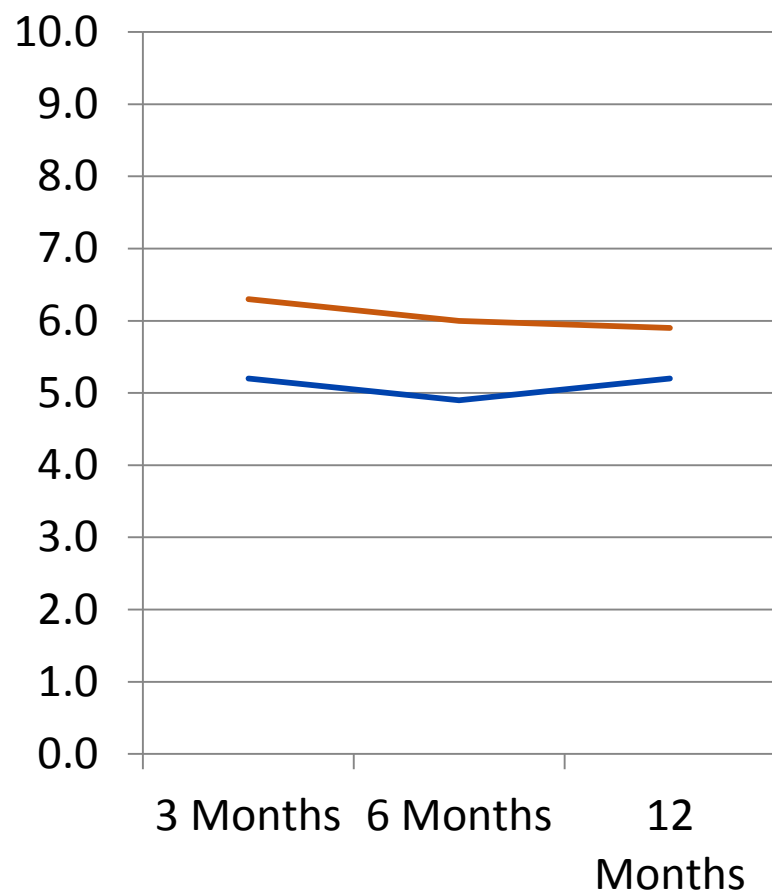
## Frequency of HA at One Year



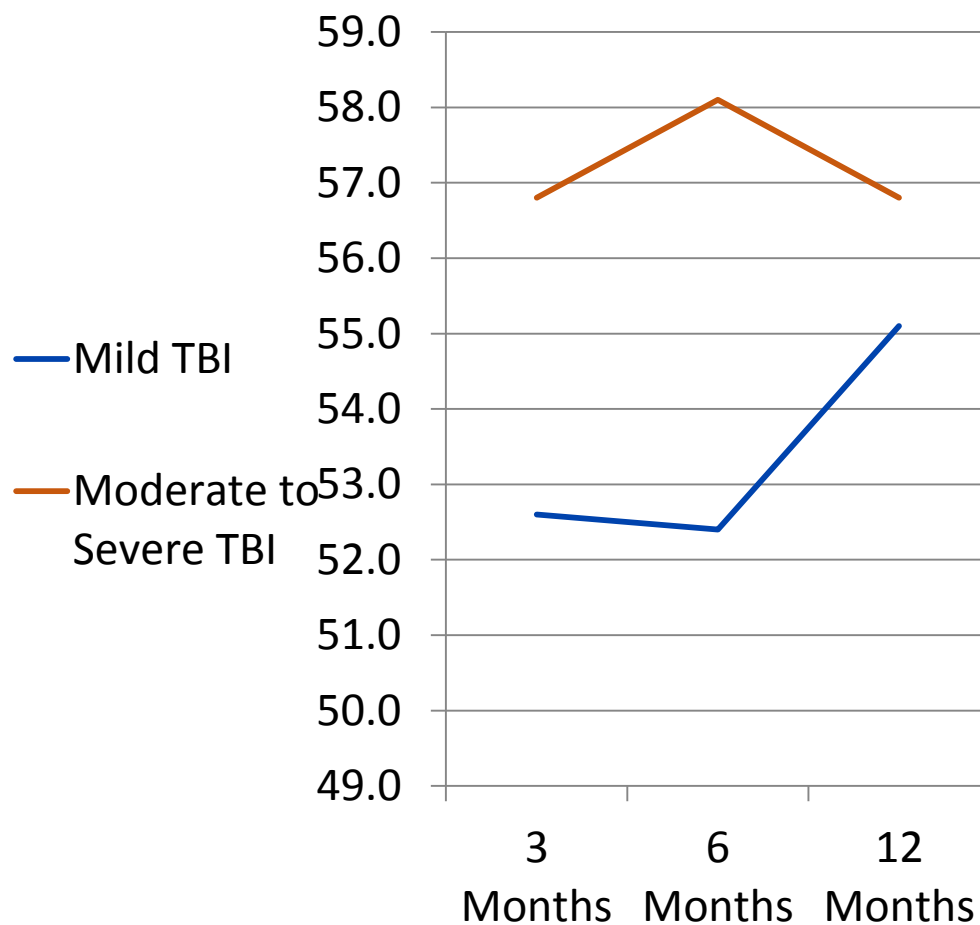


# Impact of HA

## Pain Rating

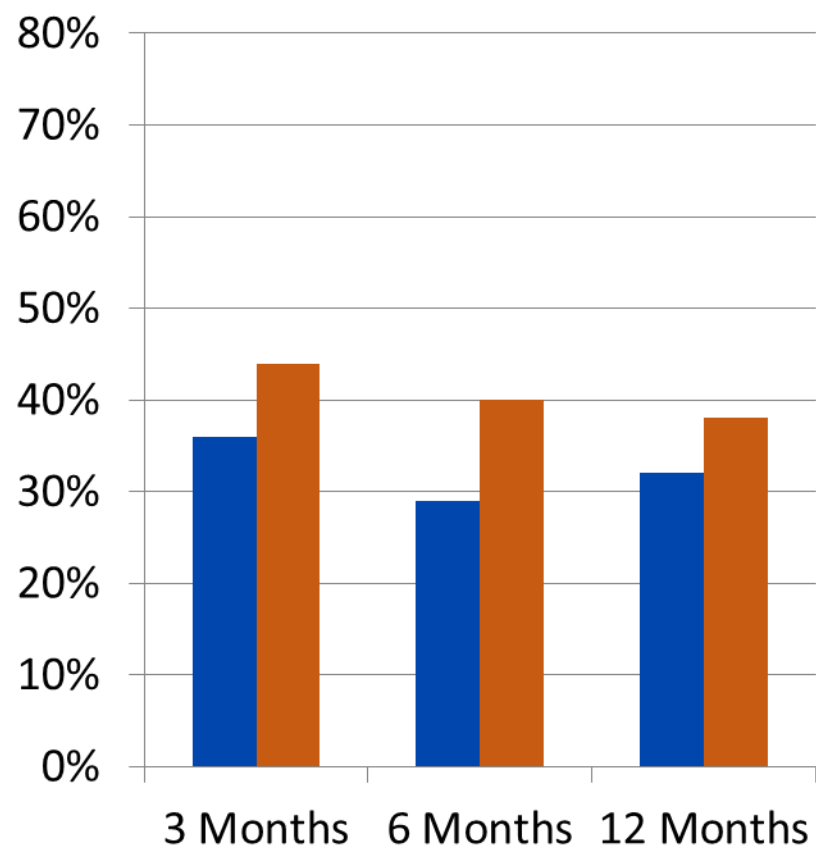


## Headache Impact Test

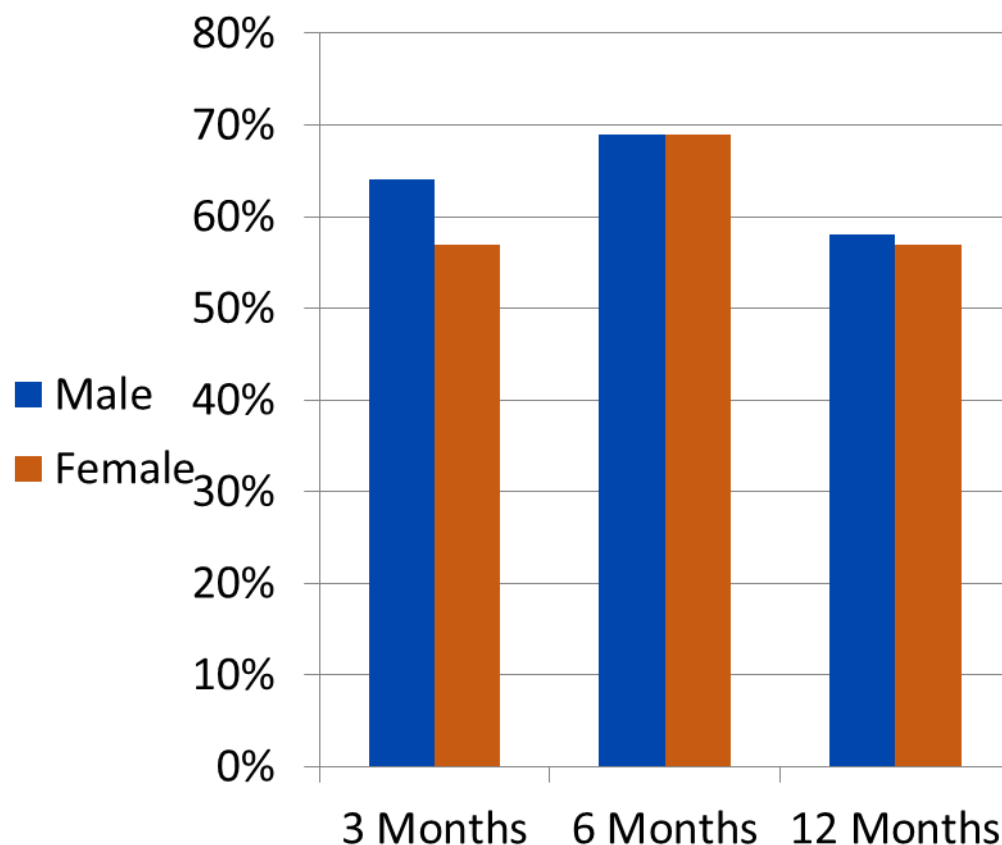


## Risk Factors – Sex

**Moderate to Severe TBI**

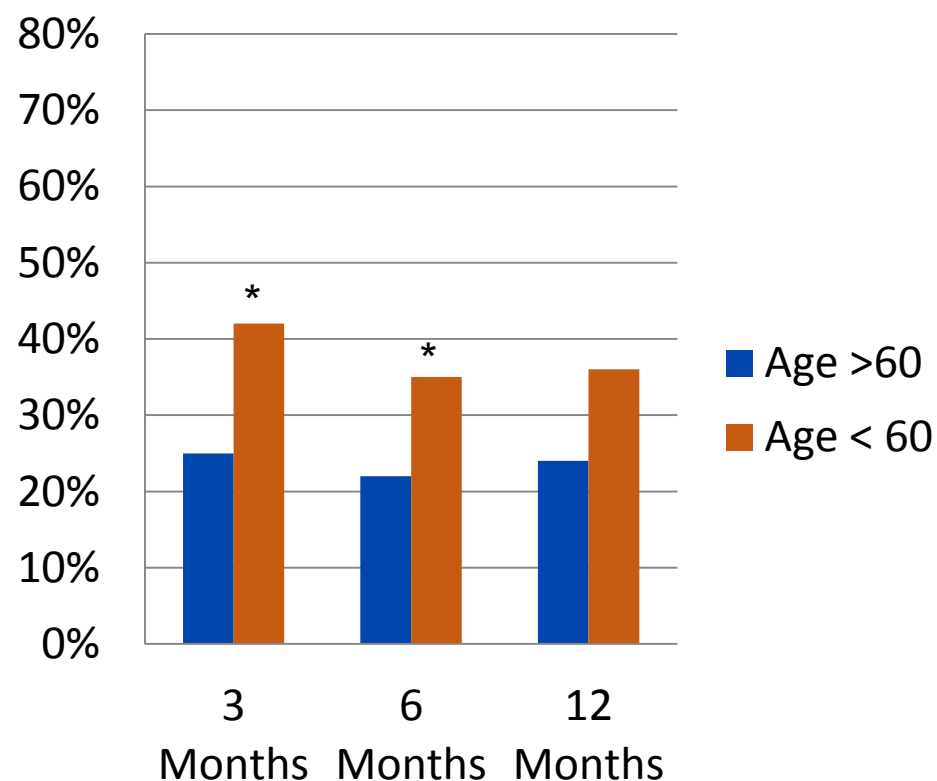


**Mild TBI**

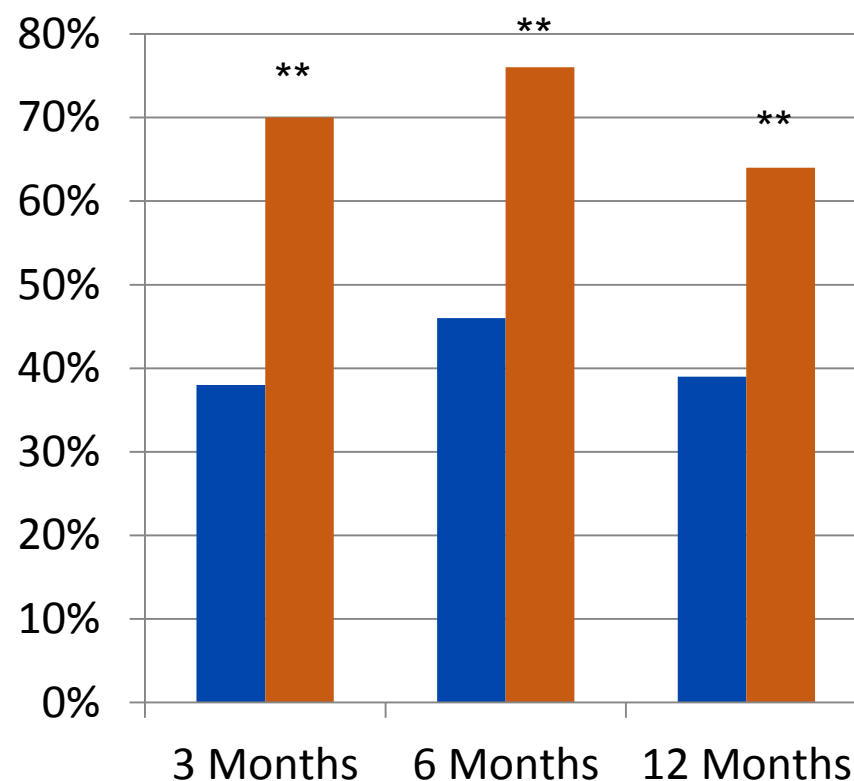


## Risk Factors – Age

**Moderate to Severe TBI**



**Mild TBI**



\* $p \leq .05$ , \*\* $p \leq .01$

## Conclusions

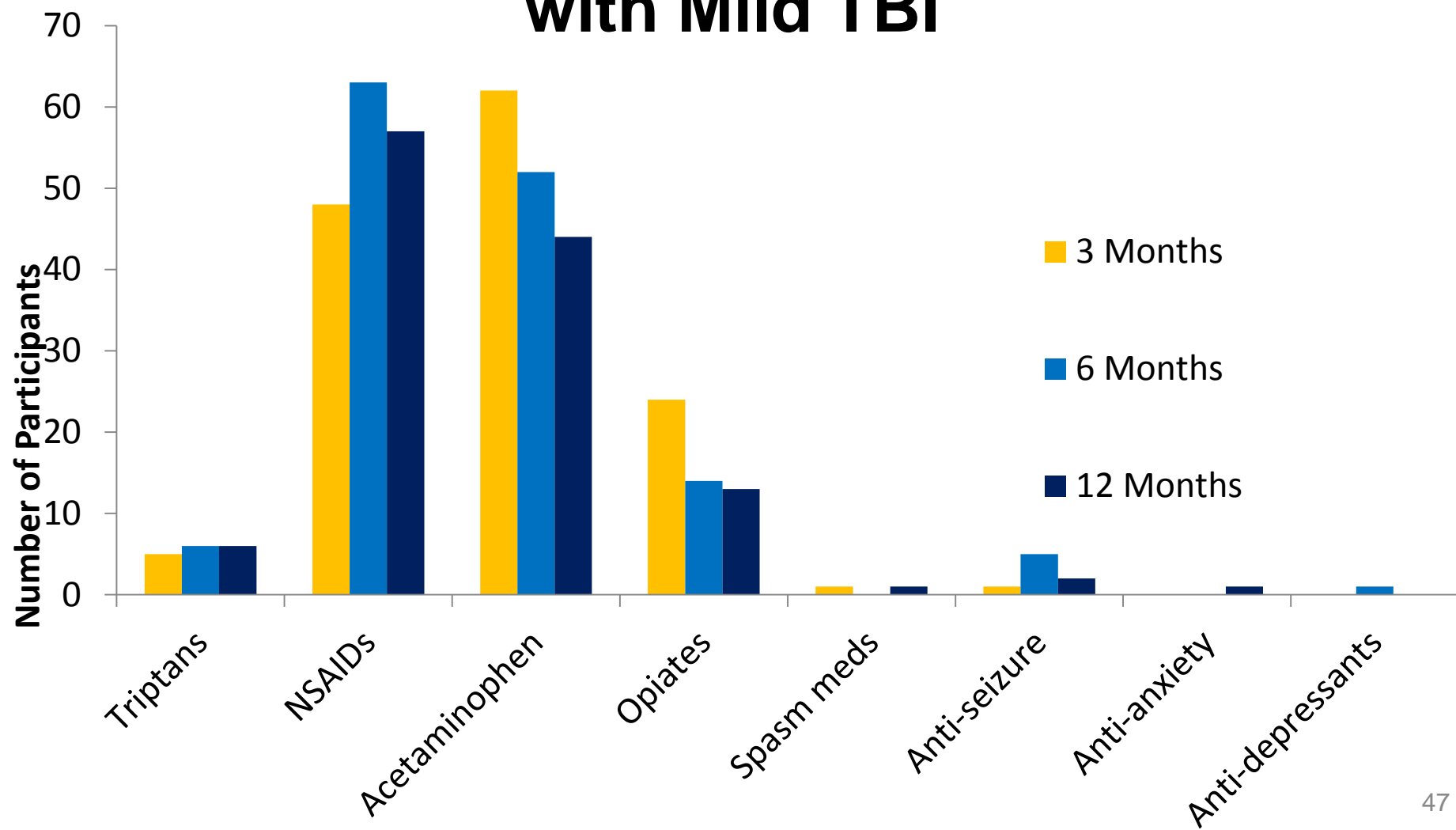
- HAs are frequent after TBI with a higher prevalence after mild than moderate to severe TBI
  - However, HA appears to be more severe in those with moderate to severe TBI
- Majority meet ICHD classification as migraine and probable migraine
- Age appears to be a risk factor for the development of HA

# Treatment of PTH

## Previous Research

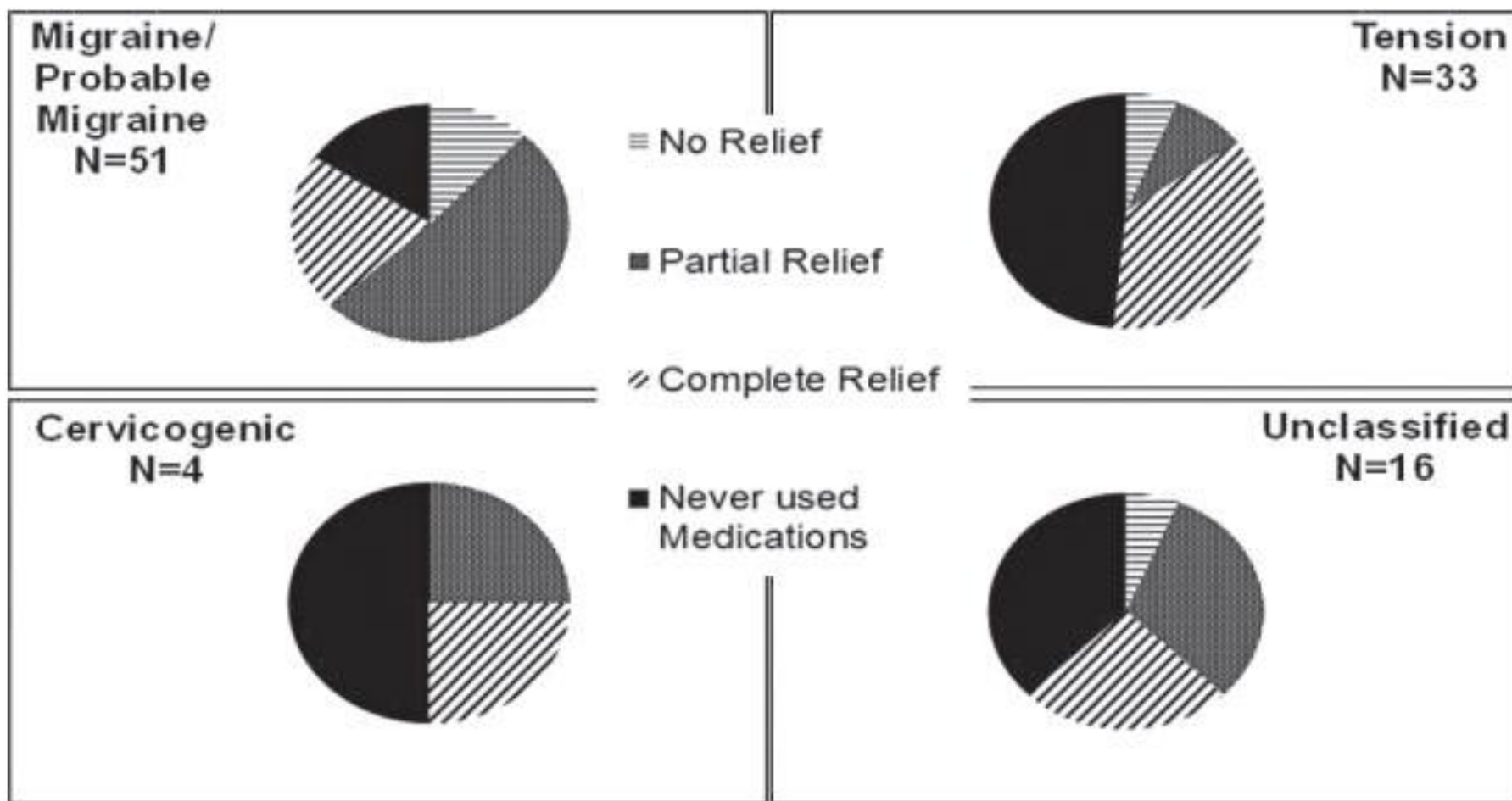
- In a review of interventions for PTH conducted in 2012 – one class II, rest III or IV
  - Pharmacotherapy
  - Biologically-based (biofeedback, physical therapy (PT), manual therapy, ice, injection)
  - Behavioral interventions (cognitive behavioral therapy (CBT), relaxation, education)
  - No evidence-based guidelines

# Self-Report of Treatment in Those with Mild TBI



(DiTommaso, Hoffman, Lucas, Dikmen, Temkin, & Bell, 2014)

# Reported Effectiveness of Medication by HA Phenotype at 12 months Post Injury





## Conclusions

- Most people with mild TBI are using over-the-counter (OTC) medications
- Very few people use alternative treatments
  - Highest (21% of those with migraine/probable migraine) use massage
- Medication does not appear to relieve PTH
- Recommendations in the literature are based on primary HA and have not been tested

# **Clinical Management of PTH**

## The PTH

- PTH criteria define the severity, latency and duration of the HA
- No **distinct clinical presentation** for PTH
  - Location is anywhere
  - Characterization of pain is variable
  - Severity can vary widely
  - Disability may be greater relative to non-PTH
  - Frequency is variable
  - Concurrent injuries such as a neck injury may complicate the clinical presentation

# Diagnostic Framework for PTH

- **Recognize** PTH following TBI.
  - Little value in acute or chronic definition
  - Latency requirement contributes to under-diagnosis (use clinical judgment past seven days after injury)
- **Evaluate** clinical features of HA.
  - Moderate to severe or disabling, location, pulsatile, physical activity makes it worse, associated features
  - Frequency may help in determining whether preventive as well as acute therapy is necessary
- **Treat** the phenotype.
- **Recognize** comorbid conditions seen with migraine.

# Differentiating a Migraine from a Tension-type HA Phenotype

## Migraine

- Moderate to severe
- Often unilateral (60%), aura in a minority of patients
- Exacerbated by routine activity
- Throbbing or pounding
- Nausea, vomiting, photophobia and phonophobia are common

## Tension Type

- Mild to moderate
- Usually bilateral
- Squeezing, vice-like, tight
- Photophobia OR phonophobia sometimes present
- No nausea or vomiting

## **Treat the Phenotype**

- Treat PTH as a primary HA disorder
  - Prior history of HA or family history of HA may make it more likely to respond to the treatment but this needs further study
- Severity of HA may determine need for non-specific or specific migraine therapy
  - Frequency may determine need for preventive therapy
- Consider severity of TBI and cognitive impairment in the individual with PTH and choose therapy accordingly

# Strategies for Migraine Management

- Treat PTH as a primary HA disorder
  - Prior history of HA or family history of HA may make it more likely to respond to the treatment but this needs further study
- Severity of HA may determine need for non-specific or specific migraine therapy
  - Frequency may determine need for preventive therapy
- Consider severity of TBI and cognitive impairment in the individual with PTH and choose therapy accordingly

# Strategies for Migraine Management

Recognize the  
headache type

Keep a diary to  
determine  
predictable  
triggers

**Acute  
treatment  
stops the  
pain**

Set realistic goals  
Provide a care plan

Individualize care and  
recognize comorbidity

**Recognize  
and avoid  
triggers**

**Preventive  
treatment  
for frequent  
headache**



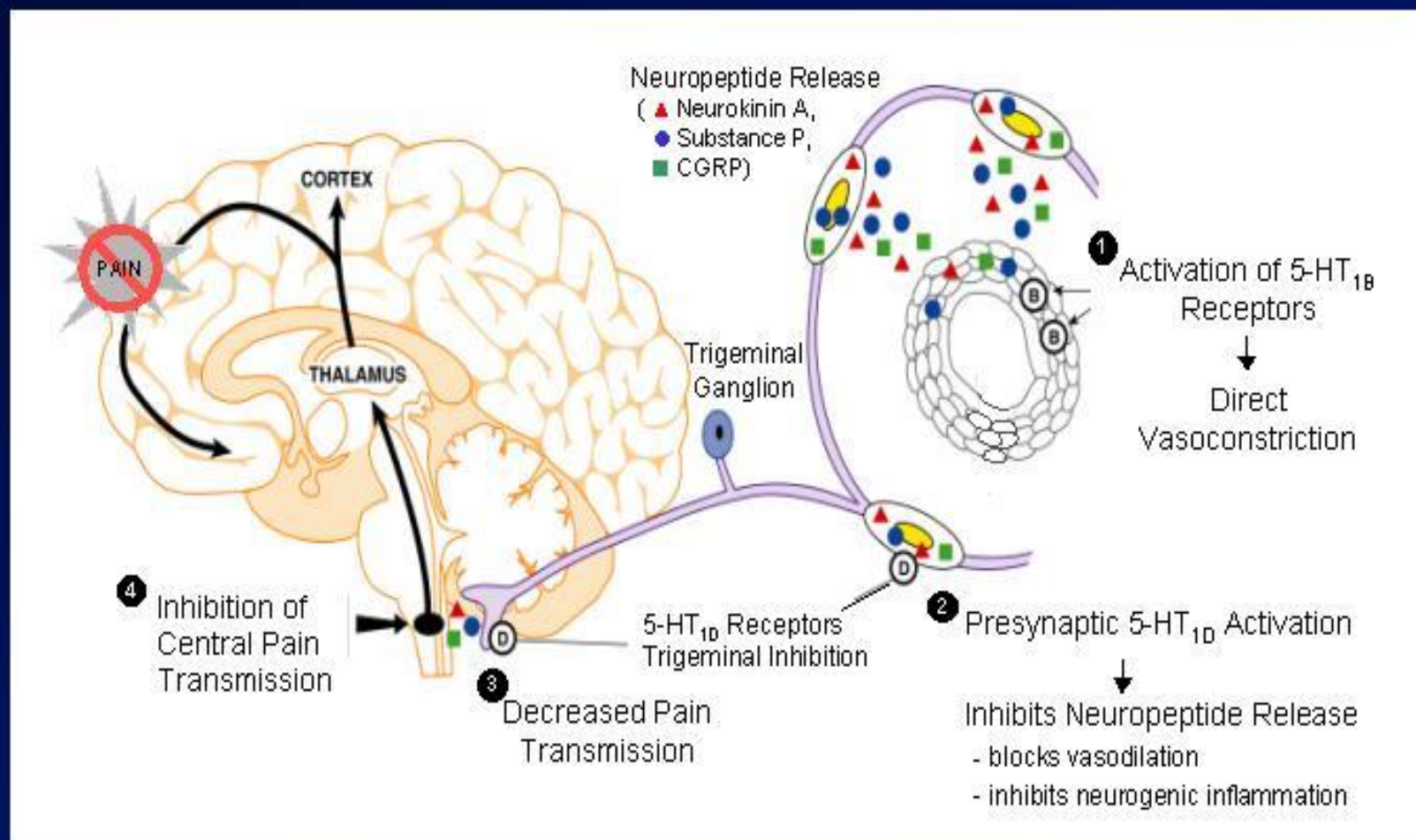
## Goals of Acute Therapy

- The goal of treatment is to restore ability to function. Stratify care based on attack severity and disability.
  - Match efficacy of initial HA therapy to the treatment need.
- Treat a migraine attack as soon as possible after onset.
  - If the HA reoccurs, re-treat.
- Minimize use of back-up and rescue medications by making sure the initial drug is effective.
  - Limit use of acute therapy to avoid medication overuse headache (MOH).
- Optimize self-care by patient education and an effective treatment plan.
- Avoid or minimize side effects by choosing medication with good tolerability as initial therapy.

# Acute Migraine Medications

- Nonspecific (can be prescription or OTC)
  - Simple analgesics
  - Combination analgesics
  - Non-steroidal anti-inflammatory drugs (NSAIDS)
  - Opioids
  - Corticosteroids
- Specific
  - Ergotamine/dihydroergotamine
  - Triptans
- Adjunctive therapies
  - Antiemetics/dopamine antagonists

# Acute Anti-migraine Targets



(Adapted from Hargreaves, Shephard 1999)

(Graphic used with permission from Merck)

# Migraine-specific Treatment Choices

( the “triptans” or ergot alkaloids)

- **Sumatriptan** (Imitrex)
  - Tablet (25, 50, 100mg)
  - Injection (6mg, 4 mg stat dose)
  - Single dose vial (6mg/0.5cc)
  - Nasal spray (5, 20mg)
- Sumatriptan 85 mg and naproxen sodium 500 mg (Treximet)
- Sumatriptan needleless injection system (6mg; Sumavel)
- **Zolmitriptan** (Zomig)
  - Tablet (2.5, 5mg)
  - ZMT (2.5, 5mg)
  - Nasal spray (5.0 mg)
- **Naratriptan** (Amerge)
  - Tablet (1, 2.5mg)
- **Rizatriptan** (Maxalt)
  - Tablet (5, 10mg)
  - Orally disintegrating tablet (5, 10mg)
- **Almotriptan** (Axert)
  - Tablet (6.25, 12.5mg)
- **Frovatriptan** (Frova)
  - Tablet (2.5mg)
- **Eletriptan** (Relpax)
  - Tablet (20, 40mg)
- **DHE-45** (Dihydroergotamine mesylate)
  - Injectable (4mg/cc)
- **Migranal Nasal Spray**
  - 4mg/cc

# Guidelines for Initiating Preventive Medication in PTH

- Frequency of HA greater than four-six per month, disability more than two-three days per month or that significantly interferes with quality of life
- Use of acute medication more than two-three times per week on average or escalating use
- Acute medications contraindicated, not tolerated or ineffective
- Use comorbid conditions to select preventive therapy
- Difference between PTH and primary HA treatment may involve cognitive changes following TBI
  - Compliance, memory, side effects of medication

# Migraine Preventive Medications

- Antidepressants
  - Tricyclic antidepressants (TCA): Amitriptyline or nortriptyline 10-50 mg
  - SSRI/serotonin-norepinephrine reuptake inhibitors (SNRI): Fluoxetine 10-40 mg/duloxetine 20-120 mg
- Cardiovascular
  - Beta blockers: Nadolol 40-120 mg/metoprolol XL 50-100 mg/propranolol LA 60-240 mg
  - Ca channel blockers: Verapamil SR 240-360 mg; amlodipine 5-10 mg
- Antiepileptic (AEDs)
  - Divalproex/valproic acid SR 250-1500 mg
  - Topiramate 75-150 mg
  - Gabapentin 300-800 mg TID
  - Zonisamide 100-500 mg

# Migraine Preventive Medications (continued)

- Dopamine antagonists
  - Chlorpromazine 25-50 mg
  - Atypicals: Seroquel 25 mg
- Other
  - NSAIDs
  - OnabotulinumtoxinA
  - Tizanidine 4-8mg or baclofen
  - Magnesium/riboflavin/feverfew
  - Memantine 20 mg BID
  - 5HT<sub>2</sub> antagonists: Cyproheptadiene 4-8 mg, mirtazapine 15-45 mg
- Nonpharmacologic
  - Biofeedback
  - CBT
  - Acupuncture
  - PT/craniosacral therapy
  - Occipital nerve block/trigger point injections

# Polling Questions

**Please rate your comfort with treating HA.**

- A. Very comfortable
- B. Comfortable
- C. Somewhat comfortable
- D. Not comfortable
- E. N/A

**Where do you seek HA diagnosis and treatment information to use in your practice?**

- A. Peers
- B. Medical journals
- C. Professional organizations, e.g., American Academy of Neurology, International Headache Society, National Headache Foundation
- D. Websites
- E. N/A



## **A Case of PTH**

- A 32 year old woman is referred to a concussion program six months after she sustained an injury during her city league soccer game. She was shouldered in the right temple by an opposing player
- She was thrown to the ground, hitting the grass turf with the left side of her head. She saw stars. She does not remember a conversation with her coach immediately after the event
- She rested on the sidelines with an ice pack to treat a HA of immediate onset. That evening she noticed a stiff and painful neck

## Next Day

- The next day she had trouble walking with gait imbalance. A local emergency room diagnosed her with a mild concussion
- She was treated by her primary care provider with PT which she thought made her HAs worse, cyclobenzaprine 10 mg up to three times a day and hydrocodone/APAP 10/325 mg up to four times a day
- She cannot work if she takes these medications so she usually takes ibuprofen 800 mg three or four times a day and the other medications at bedtime

## The HA

- HA has been constant “24-7” with severity 5-6/10 to 10/10
- Cap-like over the vertex stopping at a band around her head with bilateral orbital pain
- Allodynia over head with severe pain
- Squeezing and throbbing when severe
- Nausea and vomiting, photophobia and phonophobia
- Severe neck pain worse with computer use
- Missing about one day per week of work
- Avoiding social activities and housework, lays on the couch because physical activity makes it worse, watches TV, trouble getting to sleep

## Other Relevant History

- Has a prior history of mild HA around the time of her menstrual period
- Family history of HA: paternal grandmother, father and uncle have moderate to severe HAs
- Some recent weight loss, poor sleep and feeling anxious and unhappy
- Other medications: Oral contraceptives, vitamin D, levothyroxine

## Diagnosis?

- Chronic PTH following mild TBI with concussion
- Increases in intensity at work when working on the computer
- Evaluate clinical features of HA
  - Throbbing, physical activity makes it worse, moderate to severe in intensity, nausea/occasional vomiting, photophobia and phonophobia
- Primary HA features of migraine and comorbid conditions seen with migraine/concussion
- Decision to treat with drugs commonly used for acute and preventive therapy of migraine

## Medications

- Acute therapy for most severe HAs: Triptans with or without an NSAID
- Preventive therapy
  - Amitriptyline 10 mg at bedtime (sleep and pain blocker); alternative drug may be tizanidine
  - Fluoxetine 10-20 mg (for mood, chronicity of HA, synergistic with TCA effect)
  - Alternative therapies may be duloxetine or an anti-epilepsy drug

## **Can Any Other Diagnosis be Made?**

- Currently taking ibuprofen on a daily basis, hydrocodone/APAP and cyclobenzaprine frequently
- Diagnosis: Chronic PTH but.....
  - Chronic migraine?
  - MOH?
  - Cervicogenic HA?

## Avoid MOH

- May occur in patients with preexisting primary HA disorders
  - Anecdotal evidence for occurrence in PTH
- Pattern of HAs and overuse of analgesics in predictable and escalating frequency
  - Waking with early morning HA
- Prevention: Limit frequency of medication use
  - Ideal to keep acute treatment to two-three days per week acute medication use but do not undertreat
- Treatment: Refractory to otherwise appropriate acute and preventive therapy
  - Withdrawal therapy



# Principles of MOH

(also known as “rebound HA”)

- Taper or “cold-turkey” off medications most likely to cause MOH/rebound
- Substitute acute medications that are less likely to cause MOH/rebound (avoid caffeine-containing medications)
- Preventive program during withdrawal
  - Parenteral dihydroergotamine mesylate
  - Low-dose tizanidine with long-acting NSAIDs
  - Daily doses of a triptan for up to ten days
  - Short course of steroids, long-acting NSAIDs
- Preventive medication

## Cautions:

- Opiate and barbiturate abstinence syndromes
- Increasing HA during withdrawal period

# Common Comorbidities of Migraine

- Some comorbid conditions often found in individuals with migraine may also be symptoms of a concussion.
  - Depression
  - Anxiety
  - Social phobias
  - Bipolar disorder
  - Irritable bowel syndrome
  - Sleep disorders
  - Fibromyalgia

# Use Comorbid Conditions to Assist with Selection of Preventive Therapy

- Anxiety
- Depression
- Irritability
- Insomnia
- Somatic pain or fibromyalgia
- Raynaud's disease
- Concentration or attention difficulty



- SSRI, SNRI, AED
- SSRI +TCA, SNRI
- SSRI, SNRI, AED
- TCA, atypical, mirtazapine
- Tizanidine, TCA, SNRI
- Ca channel blocker
- CBT

# Conclusions and Future Directions

- HA is a significant problem after TBI with a large number of individuals reporting severe, often disabling HA one year following injury
- HA characterization across the entire first year after injury was most frequently consistent with migraine
- Classification of HA according to IHS primary HA criteria may assist with identifying more effective treatment for individuals with PTH
- Intervention studies are needed to determine whether HA after TBI can be treated more effectively using evidence-based guidelines and whether chronic daily HA can be prevented



## Future Directions

Photo courtesy of Sylvia Lucas, M.D., Ph.D.



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# Neuroimaging Following Mild Traumatic Brain Injury in the Non-deployed Setting

DCoE Clinical Recommendation | July 2013

## Neuroimaging following Mild Traumatic Brain Injury in the Non-Deployed Setting



### Introduction and Background

More than 273,000 service members have sustained a traumatic brain injury (TBI) between 2000 and the first quarter of 2013.<sup>1</sup> The majority of these (approximately 85%) occurred in the non-deployed environment and 82.4% were classified as mild TBI (mTBI), also known as concussion.<sup>1,2</sup> Neuroimaging following mTBI has been addressed in the deployed setting by the Joint Theater Trauma Systems (JTTSS) Clinical Practice Guideline, "Use of Magnetic Resonance Imaging in the Management of Mild Traumatic Brain Injury (mTBI) Concussion in the Deployed Setting", the "Department of Defense Guidance for Management of Mild traumatic Brain Injury/ Concussion in the Deployed Setting" (Department of Defense Instruction DoDI 6490.11) as well as the

The guidance contained in this CR represents a review of currently published literature and expert contributions obtained by the Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury (DCoE) in collaboration with clinical subject matter experts representing the Services, Department of Veterans Affairs (VA), academic, research and civilian sectors. The TBI Quad Service group, an inter-agency, multi Service collaborative effort organized by TBI subject matter experts, which includes representatives from the Army, Navy, Marine Corps, Air Force, Defense and Veterans Brain Injury Center (DVBIC), Army Medical Research and



### Clinical Recommendation:

### Neuroimaging Following Mild Traumatic Brain Injury (Non-deployed Setting)

#### Neuroimaging Recommendations following mTBI

Modality	Clinical indications in mTBI	Acute (days post injury) (CT, MRI, PET, SPECT, or functional MRI)	Sub-Acute (6-8 days post injury) (CT, MRI, PET, SPECT, or functional MRI)	Chronic (Months post-injury) (CT, MRI, PET, SPECT, or functional MRI)
CT	Only when there is a high suspicion of moderate to severe TBI.	Not recommended.	Not recommended.	Not recommended.
MRI	Maximum recommendation is a 1.5T MRI with T1, T2, FLAIR, DWI, and SWI sequences. MRI should be performed within 72 hours of injury. MRI should be performed within 72 hours of injury. MRI should be performed within 72 hours of injury.	Not recommended.	Not recommended.	Not recommended.
PET	Not recommended.	Not recommended.	Not recommended.	Not recommended.
SPECT	Not recommended.	Not recommended.	Not recommended.	Not recommended.

Red Flags and Acute Imaging Indications <sup>1,2</sup>	Between Neuroimaging Techniques in mTBI Pathophysiology
<ul style="list-style-type: none"> <li>Progressive decline in level of consciousness (LOC)</li> <li>Pupillary asymmetry</li> <li>Seizures, repeated vomiting</li> <li>Drop of Alcohol</li> <li>Coagulopathy</li> </ul>	<ul style="list-style-type: none"> <li>Progressive decline in level of consciousness (LOC)</li> <li>Clinically verified LOC &lt; 5</li> <li>Double vision</li> <li>Worsening headache</li> <li>Visible physical injury above clavicle</li> <li>Unusual behavior</li> <li>Age &gt; 60</li> </ul>
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- Provides guidance as a standard approach for imaging following mild TBI in the non-deployed setting to include:
  - When to order neuroimaging studies and other referrals
  - Type of neuroimaging indicated
  - Other clinical factors to consider

- Packaged with the clinical support tool

- Target audience: medical providers

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# Questions?

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May 22, 2014

1-2:30 p.m. (EDT)



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## **Next DCoE TBI Webinar:** ***Why Does Concussion Affect Men Differently Than Women?***

June 12, 2014

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